LABORATORY ANIMAL WELFARE WORK: UNREPORTED CRIMES AND LAWYERS

Prepared for
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HISTORICAL NOTE:

UAA was founded in 1967 to inform the public on what was being done to laboratory animals and to promote alternatives to the use of live animals in research and testing. When the tax laws changed permitting us to lobby to a limited extent, we developed the Research Modernization Act to give substance to these goals. That bill drew more public support than any issue ever except Medicare. Still, the major wealthy societies in this country would not support it; instead they rode on its back, so to speak, to put forth their own bill calling for humane treatment before and after *but not during* actual experimentation. Although it turned out they had "no data" to justify their own bill, by interceding they effectively killed the Research Modernization Act.

We realized then that henceforth we would not dare to put forth legislation dealing with laboratory animals. As a consequence, the Research Accountability Act, which, although it would save millions of animals, is not an animal welfare bill. We've been careful to point this out to legislators.

Both of our innovative bills required a staggering amount of highly technical background work. But to this day, the protagonists of humane treatment before and after...have not provided *factual evidence* of why their timid bill was needed, and have never, to our knowledge, reported consistently, convincingly, and in depth on what is done to laboratory animals *during* experimentation. This problem was dramatically illustrated in a direct mail newsletter, published by the Humane Society of the United States, received in New York on or about November 10, 1983. First, it promised the reader that, by joining the HSUS, "you will support a major thrust by HSUS to save some—and perhaps some day nearly all" of the laboratory animals that die each year. But then it pointed to the secrecy of the laboratories and trumpeted: "And we also are asking, loudly, insistently, and often, 'What are you doing in those laboratories?' " Thus, admittedly, the HSUS doesn't know.

In addition, how do you account for the incredible fact that, after we had poured out fully documented accounts of NASA's cruelty to animals along with photographs for more than 3 years, virtually the entire humane movement completely ignored this information; instead, it "united" and ran at the primate centers, concerning which, it turned out, "400 animal societies knew little or nothing." As a result, we turned over copies of a substantial amount of material on NASA to the conservative HSUS, but our feeling is that whatever the HSUS may do now is too little too late.

Ironically, what is done to animals in laboratories is NOT secret - not even most military vivisection. It is openly published in more than 20,000 journals and government reports. But the information goes unreported to the public and to Congress. How can you promise to stop something when you can't tell legislators or the public what it is?

Although Congress is likely to pass "humane treatment before and after..." measures, we have come to believe that little if anything can be done about the pain and fear of laboratory animals *during* experimentation unless the problem is addressed by forces outside the traditional humane movement, torn as it is - and always has been - by divisiveness and dissension, with consensus impossible of achievement due to lack of information. Going further, we believe the legal profession has a proper and necessary role in the defense of laboratory animals; for example, after more than a century of animal research, the word "pain" *has never been legally defined.* Pain is a basic response that serves to perpetuate the species - all species. We believe it's time for legal minds to address the continuing agony of laboratory animals; we have impressive information that can help.

UNREPORTED CRIMES

People may think that "crime" refers solely to breaking a law. Not so. The word also means evil conduct, evildoing, misdoing, corruption, evil courses, "misprision," which in turn means secrecy. Thus, the word "crime" fits very well with what is being done to laboratory animals: doing violence to living flesh and sentience.

These laboratory crimes go unreported. The exploiters of animals describe them in professional journals which the public never sees, or occasionally dress them up for the media. At the same time, the

defenders of laboratory animals do not report what is being done to the animals either, except for "one-cruelty" episodes that come to light sporatically, which legislators and others perceive as being only isolated instances. Thus, powerful legislators have asked for a study because "we don't know what the problem is or whether there is a problem." How could a judge in a courtroom (and that, essentially, is what Congress is) pass sentence on a perpetrator if he was presented with so little evidence that he didn't know what the problem was or whether there was a problem. Without substantive evidence to convict him, the perpetrator would walk out free. The exploiters of laboratory animals have been walking out free for more than a century because their crimes against the animals go unreported.

> IMPORTANT NOTE: We are not saying that *animal experimenters* are criminals. We are saying that their *experiments* are criminal in conformity with the above definitions. There's a big difference. In the early 1970's we were sued by a researcher who charged, among other things, that we had called him cruel. We never called *him* cruel. After all, we didn't know him. We called his experiments cruel. The case, with the aid of the research lobbies, was carried to the Supreme Court, with UAA winning all the way. We won because, under the First Amendment of the Constitution, if we believed his *experiments* were cruel, we had the right to say so. We believe the experiments reported here constitute crimes as defined above, that the animals used are victims of these crimes, and we herein exercise our right to say so.

THE PLIGHT OF HUMANE LAWYERS

Increasing numbers of humane lawyers are taking an interest in "animal rights," a philosophical view of overall animal welfare. They've done well in many of their cases involving animals, but so far as laboratory animals are concerned they remain virtually paralyzed, immobilized, because the laboratory crimes go unreported. They have no evidence to work with, although animal research is increasing explosively; even as this report is being prepared, Bausch & Lomb, a manufacurer of optics, is negotiating to acquire the shares of Charles River Breeding Laboratories because it thinks "there are excellent future prospects" for profits.

NEW LEGAL PRECEDENTS NEEDED

We believe not only that a great many experiments constitute crimes against the animals, but also that the laboratory animal users themselves recognize this. They long ago succeeded in exempting laboratory animals from state anti-cruelty laws, since there is no way they could conduct much of their research under those laws without risking liability for legal crimes. Their justification is NOT that their experiments *are not cruel*, but that they do not "intend" them to be cruel - they do not intend "wanton" cruelty. Nevertheless, they *do* intend, in conceiving the experiments, to do violence to living flesh and sentience.

Herein are random samples of experiments which we believe constitute crimes against laboratory animals. The generally accepted reasons for committing such crimes are "to save human lives" or "for the greater good." We do not deal with the reasons; everybody who perpetrates a crime has a reason.

We invite humane lawyers, in or out of the animal welfare movement, to give serious consideration to the possibility of establishing *legally* the concept of, or precedent for, crimes against laboratory animals as defined above. A number of such lawyers will be reading this newsletter, and we will take it up with others. Our members and friends also might wish to discuss this subject with humane lawyers of their acquaintance.

In the case of independent lawyers or law firms who have an interest, we invite proposals as to any action they might wish to initiate, even experimentally, on behalf of the victims of crimes in the laboratories. In the event they can offer proposals of interest to us, we would then be willing to initiate a Laboratory Animal Legal Defense Fund for their use. Lawyers who represent, or serve on the staffs of, animal welfare societies, would, of course, have access to their own financial resources. We will make available our full background data to any lawyer(s) who are able to make proposals acceptable to us.

It is our hope, too, that this exposition may be helpful to those humane philosophers who have sought to defend laboratory animals but have been forced to harangue about "morals and ethics" because the crimes go unreported.

The following crimes as defined herein were not perpetrated in the dark ages, nor even ten or twenty years ago. They are being committed *now*, in the 1980's, and supported largely by government funds -your funds. The information comes from our files and from documents that routinely flow into our office. No extraordinary search was conducted. It should be noted that 90% of the animals being used today are the little laboratory rats and mice, who suffer just as acutely from pain and fear as do dogs, cats, rabbits and monkeys. They're not even counted under the Animal Welfare Act. The experiments decribed below are not isolated instances. In almost all cases, they are part of a pattern. Also NOTE that they involve *research,* not product safety testing.

Members and friends will observe that, for the first time, we have not identified literature sources or institutions. The reason is that we don't want to instigate any spurious legislation based on this information, nor to provoke any "one cruelty" or "one institution" activity which legislators and others perceived to be "isolated instances."

Descriptions appear on the following pages.

Description	Type of Institution	Source of Funds
Repeated Separation from Cloth Mother Monkeys - 1982 In Phase 1, 6 squirrel monkeys were separated from their mothers at age 5 days and placed with "cloth mother surrogates" until the monkeys were 3 months old. Then a "repeated separation procedure" was begun, in which the surrogates were removed from the infants' cages for 5 days, then put back for 5 days. This procedure was continued until the monkeys were 1 year old. The animals' behavior was observed, during both separation and reunion periods, as follows. "Self-directed" behaviors included huddling, self-clasping "self-orality" (sucking various parts of its own body) and self-aggression. "Mother-directed" behaviors (observed only during reunion periods) included touching the surrogate, "mounting" (climbing on and clinging to the surrogate with the hands and feet), aggression toward the surrogate (biting, hitting and pulling), and "social" exploring (looking at or investigating the surrogate). Other behaviors included screeching, pacing, exploring the cage, and "hyperactivity" ("extremely rapid or vigorous jumping or leaping"). The animals showed significantly more hyperactive behavior and exploring during separation periods ("protest response" to separation) than during reunion periods. In Phase 2, the surrogates were removed for 1 year, following which they were again returned to the animals' cages. "Identical cloth surrogates" were also placed with normally-reared monkeys which were used as controls. All monkeys were allowed "20 days of constant access" to their surrogates, then the "repeated separation procedure" was begun again and continued for 30 days. Hyperactive behavior in surrogate-reared animals during separation was greatly increased in Phase 2 compared to Phase 1, indicating that the "protest response" to separation was stronger after the monkeys have been deprived of the surrogates for 1 year. Other behaviors seen during both separation and reunion periods, which were greatly increased in Phase 2 compared to Phase 1,included self-clasping, head clasping, self-aggression, surrogate-directed aggression, pacing, and screeching. None of these behaviors, except pacing and screeching, were seen in the controls. The experimenters concluded that "the [intial] 1-year social isolation and [repeated] surrogate separation resulted in an intensification of...abnormal behaviors."	university	not acknowledged
Social Deprivation Disrupts Sexual Behavior in Monkeys - 1982 The sexual behavior of 5 laboratory-born, ovariectomized (ovaries removed) female rhesus monkeys was compared to that of 5 wild-born female monkeys, also ovariectomized, captured at 2 years of age. Eight "sexually vigorous" adult males were used as mates. The laboratory-born monkeys were separated from their mothers at 3-4 months of age, and kept in individual cages with only very brief interactions with peers each day ("moderate social restriction"). They were released into the colony at age 11 months, then again isolated at	primate center	National Institutes of Health

Description	Type of Institution	Source of Funds

age 4 years. The wild-born monkeys were captured at 2 years of age, placed in quarantine for 15 months, then placed in individual cages until they were 6. Both groups of animals were then ovariectomized and the experiment began 1 month following surgery.

First, all females were injected with estrogen (female sex hormone) for 15 days, then placed one at a time with 2 different males for 10 minutes, or "until the male ejaculated." This was repeated 2 months later using the same males. Laboratory-born females showed decreased incidence of "presenting" the genitals in response to male contact. As a result, the males displayed a greater frequency of "sexual behavior," including approach, contact, mounts and intromissions, when paired with the wild-born as compared to the laboratory-born females. The experimenters concluded that sexual behavior is "seriously impaired by even moderate social deprivation in infancy."

Pavlovian Classical Conditioning - 1982 — hospital — Veterans Administration

Previous experiments had shown that when electric shock is repeatedly paired with a tone signal, the decrease in heart rate/blood pressure normally seen at the onset of the shock can be elicited by the tone signal alone (Pavlovian classical conditioning of heart rate/blood pressure decrease). In the present experiment, 123 rabbits were used to observe the effects of partial or total ablation of the septum (an area of the brain) on this Pavlovian classical conditioning of the heart rate/blood pressure decrease in response to electric shock.

Prior to classical conditioning, various parts or all of the animals' septa were electrically ablated. Other rabbits were sham-ablated (skulls surgically opened and electrodes inserted but no current passed) and used as controls. At the same time, other electrodes were implanted into the hippocampus (another brain area) of some of the brain-ablated and sham-ablated rabbits to record hippocampal brain waves. After recovery from surgery, the classical conditioning was begun. First, a cannula was implanted under local anesthesia into an ear vein to record blood pressure. "Stainless steel safety pins" were inserted into the right front leg and left haunch to measure heart rate. "Insulated insect pins" were inserted into a neck muscle to measure electrical activity in the muscle by electromyography (EMG). The rabbits were then immobilized in "standard plexiglas rabbit restrainers," and presented with tones of either high or low intensity followed by electric shock to the eyelids through "chronically implanted stainless steel wound clips " to observe the effects on heart rate, blood pressure, hippocampal brain waves, and EMG. For half the animals, high intensity tone signalled shock and low intensity tone signalled no shock. For the other half, low intensity tone signalled shock, while high intensity tone signalled no shock. All animals received two sessions of 128 electric shocks each, resulting in lowered heart rate and blood pressure during presentation of tones signalling shock, as seen in previous experiments. Heart rate and blood pressure were lowered even further in animals with lateral or

Description	Type of Institution	Source of Funds

complete septal ablation compared to animals with ablation in other areas of the septum or sham-ablated animals. Brain wave activity was disrupted in animals by medial septal ablation but not by lateral septal ablation. No heart rate/blood pressure changes were seen during the tones which did not signal shock.

Following classical conditioning, the animals received 7 daily sessions of unsignalled, random electric shocks of varying intensities to the eyelids to observe the effects of septal ablation on "shock threshold" (intensity of shock) which produces "twitch," "eye-blink," and "jump response." The thresholds obtained were "highly similar to those reported in a previous article," and were not affected by partial or total septal ablation. Finally, the animals were placed in an "open field box" once each day to observe the effects of septal ablation on "locomotor behavior" (activity). Rabbits with complete septal ablation showed increased motor activity, while animals with partial septal abaltion or sham ablation showed decreased motor activity. All animals, including sham-ablated controls, were killed at the end of the experiment to verify brain area ablated.

Air Blast: An Alternative to Electric Shock in Operant Conditioning - 1982

Research and Education Foundation

National Institutes of Health

5 wild-born baboons were used in an experiment to produce a "modified approach" to the operant conditioning of elevated blood pressure. First, a catheter was implanted through a leg vein into the aorta (heart·artery) and connected to a device located in a "lightweight fiberglas backpack" to allow measurement of heart rate and blood pressure in the animals' cages. Another catheter was implanted into the vena cava (heart vein) to take blood samples. The animals were allowed to recover from "catheter implant surgery," then operant conditioning was begun. The animals' resting (baseline) blood pressure was measured, and used as "initial criterion pressure." Each time the animals' blood pressure rose above "criterion pressure" a green light came on, and a timer was activated. When the animal had accumulated 120 seconds of "high pressure time," it received a food reward. However, when the animals' blood pressure fell below "criterion pressure" a red light came on and another timer was activated. After 120 seconds of "low pressure time" the animal was punished with an air blast delivered through a set of 10 "orifices" placed in the animals' cages in such a way that they "could not escape a direct blast of air from at least one orifice." Operant conditioning continued for 8 hours each day, 7 days a week, and each day the "criterion pressure" which the animals had to maintain to avoid the air blast was increased, forcing the animals "to maintain increasingly higher levels of [blood] pressure in order to maximize the number of rewards and to minimize the frequency of punishment." Blood samples were taken periodically during daily operant conditioning to observe plasma cortisol levels to assess stress.

Following daily sessions of operant conditioning extending up to 80 days, the animals showed increases in blood pressure "ranging from 17.5% to more than 44%." Heart rate was also increased, "from

Description	Type of Institution	Source of Funds
7.3% to more than 50%" higher than pre-conditioning heart rate. Blood samples analysis showed increased plasma cortisol levels, indicating that "a generalized stress response may play a role in the induction and maintenance of conditioned hypertension." The experimenters concluded that air blast can be used as an "alternative" to electric shock for operant conditioning of elevated heart rate and blood pressure.		
Effect of PCP on Sleep-Deprived Aggression - 1982 One of the standard methods of depriving animals of sleep is the flower pot method. In this method, the animal is placed on an inverted flower pot in a bucket containing several inches of water, so that every time the animal falls asleep, it falls off the flower pot into the water. To produce an animal model of aggression, 70 rats were deprived of REM (rapid eye movement) sleep for 3 days by the flower pot method. Other, non-sleep-deprived rats were kept in cages for 3 days. All rats were then injected with PCP (phencyclidine, a popular street drug called "angel dust"), and the sleep-deprived rats were paired with non-sleep deprived rats. Each pair was placed in the same cage to observe the effects of PCP in aggressive (sleep-deprived) versus non-aggressive animals. The fighting included aggressive (threatening) postures, defensive (retreating or submissive) postures, and attack. Other rats, either sleep-deprived or non-sleep-deprived, were also injected with PCP. Each individual rat was then placed in a cage with a mouse, to observe the effects of PCP on mouse-killing in aggressive (sleep-deprived) versus non-aggressive rats.	university	National Institute on Drug Abuse
Drugging/Cocaine Seizures - 1983 Seven female cats were surgically implanted with 8 recording electrodes in various brain areas, and catheters surgically inserted into the animals' jugular veins. Following recovery from surgery, the cats were placed in the "experimental cage" and brain waves were recorded for 4 minutes through the previously implanted electrodes. Then the animals were injected through the cannula with either physostigmine, atropine, apomorphine, or pimozide (drugs which either enhance or block the effects of the brain chemicals acetylcholine and dopamine), or saline, and after a short interval brain waves were recorded for 4 more minutes. Finally, the animals were injected through the catheter with doses of cocaine found to be convulsive in previous experiments, and brain waves were again recorded for 5 minutes. Injection of physostigmine or apomorphine prior to cocaine injection produced increased "behavioral arousal" including pacing the cage and "investigative" sniffing and searching. Injection of pimozide, atropine, or physostigmine prior to cocaine injection resulted in brain wave changes. One minute after cocaine injection, the animals displayed "rapid orienting head movement," tremors, loss of coordination, and limb stiffness. The cats then went into convulsions, which lasted 4 minutes after cocaine injection. An abundant number of high voltage, abnormal brain waves called spindles were observed simultaneously with appearance of seizures. Atropine	university	not acknowledged

Description	Type of Institution	Source of Funds
injection prior to cocaine administration caused increased duration of cocaine-induced seizures, but reduced the number of spindles. Physostigmine reduced seizure duration, but increased the number of spindles observed. Apomorphine and pimozide had no effect on cocaine-induced seizures or brain wave changes.		
Effect of TMT on Brain-Shock-Induced Seizures - 1982 First, electrodes were surgically implanted into the brains of 205 rats, and low intensity electric shocks delivered to their brains to observe the electrical "after-discharge" (AD). Shock intensity was increased stepwise until an AD of 6 seconds was obtained (electric shock level was at 6-second AD threshold). Those rats which did not show ADs were assumed to have mislocated electrodes and were "discarded." The remaining rats were divided into three groups. The first group was given 6-second AD threshold (low intensity) brain shocks. The second group was given repeated, high-intensity brain shocks until seizures occurred (kindling). The third group was injected with pentylenetetrazol (a convulsant) to artificially produce seizures. Then all rats were injected with either saline (controls) or various doses of trimethyltin (TMT, a tin compound widely used in agriculture and industry) to observe its effects on ADs, and on kindled (electrically-induced) and pentylenetetrazol-induced seizures. TMT had no significant effects on ADs, but did cause seizures not seen in control animals. Kindling occurred more rapidly in TMT-exposed rats than in controls, and pentylenetetrazol-induced seizures were more severe following TMT injections. All rats, including saline controls, were killed at the end of the experiment to verify electrode placement.	government - non-military	U.S. Environmental Protection Agency
Nerve Poisoning - 1982 258 rats were administered various doses of trimethyltin (TMT, a tin compound widely used in agriculture and industry), to determine the LD_{50}. Some of the rats were also immobilized in "commercial restraint tubes" and temperatures sensors inserted into their rectums to observe TMT-induced body temperature changes. In surviving animals, TMT produced weight loss, decreased body temperature, "tail chasing and mutilation," vocalizing, seizures, rearing, "running fits," tremors, and violent aggression when picked up by the experimenter. All surviving rats were killed at the end of the experiment to observe TMT-induced brain damage. "Normal" controls, who were not given TMT, were also killed.	government - non-military	U.S. Environmental Protection Agency
Animal Model of Epilepsy - 1983 Lithium chloride is a drug "widely used...in the treatment of manic depressive illness" and known to cause brain damage, tremors, and convulsions in humans. Previous animal experiments had shown that lithium-induced seizures may be the result of increased sensitivity to the brain chemical acetylcholine. Thus, in the present experiment, rats were injected with lithium chloride, then 24 hours later injected with pilocarpine or physostigmine (2 drugs which enhance the effects of acetylcholine). Other rats were injected with only pilocarpine or physostigmine. A third group of rats were injected with lithium, then atropine (a drug which inhibits the effects of acetylcholine), followed by pilocarpine. Some of the rats from each	medical school	U.S. Public Health Service

Description	Type of Institution	Source of Funds
group were then "decapitated and the heads were immediately frozen in liquid nitrogen" to observe brain chemical changes. The remaining rats were observed for 4 hours for behavioral changes. Rats treated with lithium alone appeared "mildly sedated" but showed no other behavioral changes. Animals which received pilocarpine or physostigmine alone shed bloody tears, salivated, and their fur stood on end. Animals which received lithium in addition to pilocarpine or physostigmine showed these same symptoms, along with staring, repetitive mouth movements, "head bobbing," blinking, and "wet dog shakes," culminating in "seizures in which the rats reared up on their hind limbs, with their forepaws and head in clonus [jerky spasms]." The seizures lasted 30-40 seconds, and recurred every 2-5 minutes throughout the 4-hour observation period. Seizures were not seen in those lithium-treated animals which received atropine prior to pilocarpine injections. Following behavioral observation, the animals were killed for autopsy. Examination of the animals' brains revealed extensive damage in a number of brain areas. No damage was seen in those rats given atropine injections prior to pilocarpine injections. The experimenters concluded that lithium-induced seizures may provide "a valuable animal model for studying epilepsy."		
Punishment by Nicotine - 1983 "The possibility that nicotine...might function as a punisher to suppress behavior has not been explored." To investigate the punishing effects of nicotine, squirrel monkeys, which had previously been studied with a "variety of drugs," were starved to 80-85% of their normal weight. A catheter was surgically implanted in a vein, and the monkeys were encased in "leather jackets" to protect the catheter. They were restrained in chairs, and some monkeys had their shaven tails restrained in a "small stock" with electrodes attached to the tail for delivery of shock. A green or red light signalled that lever pressing would produce food. Then the procedure was changed so that the red light signalled that lever pressing would be punished by an electric shock or, when paired with an amber light, punished by an automatic injection of nicotine through the catheter. Nicotine doses and electric shock intensity were increased stepwise until the monkeys refused to press the lever in the presence of either the red light or the red and amber lights to avoid punishment (electric shock/nicotine). High doses of nicotine caused some of the monkeys to vomit. The researchers then injected them with chlordiazepoxide (Librium) or mecamylamine (a nicotine antagonist). After injection of the tranquilizer (Librium), the monkeys resumed lever pressing in the presence of the red/red and amber lights, even though they were punished by electric shocks or nicotine. Mecamylamine, which antagonized the effects of nicotine, caused the monkeys to resume lever pressing in the presence of the red and amber lights because nicotine no longer punished them, but they refused to press the lever in the presence of the red light alone to avoid electric shock punishment. The researchers concluded that nicotine is a punisher for monkeys.	primate center	U.S. Public Health Service

Description	Type of Institution	Source of Funds
Learning in the Isolated Brain - 1983 To study learning in the intact brain isolated from the body, rats were anesthetized with ether and their spinal cords severed at the base of the brain in accordance with the method developed in France in 1936, in order to produce an alert, isolated brain (encephale isole) and a paralyzed body. The animals were placed in the stereotaxic instrument to immobilize the head. After 1-2 hours, "to allow for recovery...from surgical procedures, and after whisker twitches and eye movements began to occur, indicating alertness," a bite hose was placed in the rat's mouth. A local anesthetic was applied to the animal's palate, jaw, and other oral tissues to prevent any unprogrammed pain from the hose or the stereotaxic instrument, "since such stimuli produce biting." "Following placement of the bite hose and confirmation of local anesthetization, electrodes were implanted" into the brain of the conscious animal, in areas where electrical stimulation is known to be rewarding (pleasurable). Electric shock was delivered to the brain, with current intensities increased stepwise until "whisker twitching and sniffing responses were elicited," confirming that the brain stimulation was rewarding. If no response was observed, the electrodes were lowered deeper into the brain and testing was repeated. If the electrode reached the base of the brain and the desired response was still not observed, the electrode was removed and reimplanted elsewhere in the brain. One control animal (non-spine-severed) was permanently implanted with electrodes, and testing was begun 14 days following surgery. The animal was placed in the experimental chamber and electric shocks of increasing intensity were administered to the brain until "whisker twitching, sniffing, increased locomotor activity and advancement in a forward direction" was obtained, indicating that brain stimulation was rewarding. The animal was then trained to press a lever in the presence of a light signal to receive rewarding brain stimulation in order to observe whether the rat could learn to discriminate between light on and light off by pressing the lever during light on, but not during light off. The encephale isole rats were given a series of rewarding brain shocks, to observe whether termination of the pleasurable stimulation would result in uncontrolled biting. "When no uncontrolled biting had occurred for a least 10 minutes," the animals were trained to bite the hose in the presence of the light signal to receive pleasurable brain stimulation. This was accomplished by the experimenter raising the rat's lower jaw against the hose when the light was on. Once biting was initiated, current intensity was adjusted, if necessary, to maintain steady rates of biting, and "biting was allowed to continue for 100-600 responses" in the presence of light. Then, to observe whether the encephale isole rats could learn to discriminate between light on and light off, the signal light was turned off, so that hose biting during light off periods did not result in rewarding brain stimulation. In some rats, discontinuation of the pleasurable brain stimulation in response to hose biting when the light was off resulted in "an initial increase in the frequency and intensity of biting." Occasional "uncontrolled biting," whether the light was on or off, was sometimes observed, and was accompanied	university	National Institute of Mental Health

Description	Type of Institution	Source of Funds
by seizures in the brain "produced by [brain] stimulation." After a time, the rats learned to discriminate between light on and light off (biting decreased when the signal light was off). The experimenters concluded that learning does occur in the isolated brain, just as it occurred in the control rat. At the end of the experiment, all rats, including the control, were killed to verify electrode placement.		
Aggression in the Isolated Brain - 1980 A researcher used two methods to study aggression behavior induced by electric shocks to different areas of the brain. In the first, electrodes were implanted and shocks delivered to 3 brain areas in the hypothalamus (perifornical, lateral, and posterior nucleus) of rats to produce "mouse biting attack and killing." Then, to compare methods, the same rats were used as encephale isole ("acute") preparations in hose-biting attacks produced by the same stimulation. Encephale isole "preparations" are created by a method developed in France in 1936, in which the spinal cords of animals are severed at the base of the brain to produce an alert, isolated brain and a paralyzed body. In the present experiments, bite hoses were placed in the mouths of the animals, and brain shocks delivered to observe the frequency and intensity of biting attacks on the hose in order to analyze "specific hose biting patterns produced by the same stimulation" which had previously resulted in mouse biting and killing. The researcher concluded that there was a "close correspondence between...the presence and degree of mouse attack and hose biting." Other encephale isole rats, with electrodes implanted in additional brain areas, were brain shocked to compare biting differences between these rats and encephale isole rats with electrode placement in the hypothalamus areas.	university	not acknowledged
Strap Biting - 1983 Electrodes were surgically implanted into rats' brains for the delivery of electric shocks to their brains, then the animals were placed in "restraint tubes" with their tails secured to "tail rods." The animals were given brain shocks, followed by a tail pinch with enough force to produce "strong vocalization." This was repeated every minute, using electric shocks of increasing intensity, until analgesia was produced by the brain shocking (vocalizations no longer elicited by tail pinch). If the brain shock did not produce analgesia, or the animal suffered seizures, the testing for that animal was ended. 24 hours later, the rats were again restrained in the tube with a nylon "biting target" in front of them, and given electric tail shocks severe enough to cause the animal to bite on the strap. The rats were then given brain shocks/tail shocks to observe the effects of brain shock-induced analgesia on the frequency of strap-biting. All animals were then killed and their brains examined to verify placement of the electrodes.	university	University Faculty Research Grant
Effects of Tail Shock on Target-Biting - 1983 Immobilized mice were used in 2 experiments to observe the effects of tail shock on target-biting in "aggressive" animals, since immobilized mice have "an initially high baseline level of target-biting" in the absence of electric shock. In experiment 1, the mice	university	University Research Council Grant

Description	Type of Institution	Source of Funds
were immobilized in narrow plastic cylinders with their tails "passed through a slot at the rear of the cylinder and taped in position." Two electrodes were attached to each animal's tail, and a bite target ("model 278-1631 cable ties, Radio Shack") placed near the animal's mouth. Twenty-minute target-biting sessions were then conducted, 5 days per week for 4 weeks. During the first week, no electric shock was delivered. During the next 3 weeks, tail shock was delivered every 2 minutes during each 20 minute session. Levels of target-biting observed in the absence of shock (first week) and during the intervals between shock (second-fourth week) were "comparatively high compared to that reported for rats and squirrel monkeys." Tail shock resulted in increased target biting immediately following shock. Experiment 2 was identical to experiment 1, except that each shock was preceded by a tone signal. As in experiment 1, tail shock resulted in increased target-biting. However, target-biting decreased sharply during the tone signal, and a "tendency of the mice to "freeze" in anticipation of electric shock was observed.		
In a continuation of the above experiments, mice which had previously been housed individually (isolated) or in groups of 4 were immobilized in plastic cylinders with their tails "passed through a slot at the rear of the cylinder and taped in position." A bite target ("Catalog No. 278-1631 cable ties; Radio Shack") was placed near each animal's mouth, then 20-minute target-biting sessions were conducted, 5 days per week for 2 weeks. Number of target-bites of previously isolated mice was greater than in mice which had been group housed. The experiment was then repeated using rats. One rat died during the experiment "for reasons unrelated to the procedure." In surviving rats, the number of target bites observed was greater in isolated rats than in group-housed rats.	university	University Research Council Grant
Learned Helplessness Reduces Aggression - 1982 180 male and 30 female rats were used to observe the effects of inescapable electric shock ("learned helplessness," in which the animals give up all hope of escaping from shock) on aggression by the dominant male of a rat colony toward an intruder. First, 60 male and all 30 female rats were assigned to colonies so that 1 female and 2 males were placed together in a cage. The remaining 120 males were used as "intruders." Females were included in the colonies because earlier, similar experiments had shown that "male dominance and aggression are faciliated by having mixed-sex colonies." Pups born during the experiment were "removed from the colony." After 8 weeks, an intruder was placed into each colony for 10 minute "sparring sessions" once each day for 4 consecutive days. These "sparring sessions" had been shown in previous experiments to be important in "increasing aggression and establishing clear dominance in one of the male residents of the colony." Twenty-four hours after the last "sparring session," another intruder was placed into the colony, and aggression by the dominant male recorded for 10 minutes as follows: number and duration of attacks, number and location of bites, time-to-piloerection (fur standing on end), and number and duration of "on-top-of instances," in which dominant	university	Mellon Foundation

Description	Type of Institution	Source of Funds
rats stood directly over intruders lying on their backs. Defensive behavior of the intruder was observed as follows: "fleeing," "defensive boxing," and "on-the-back instances." After the "colony-intruder tests" were completed, 20 of the 30 dominant males were subjected to electric tail shock in a "wheel-turn box." 10 of these rats could escape from shock by turning the wheel. The other 10 rats were "yoked" to the first group to receive simultaneous but inescapable shocks. The remaining 10 rats were not shocked. Following 80 electric shocks, all dominant males were returned to the colonies, and the "colony-intruder tests" were repeated at 24 and 96 hours post-shock, using different intruders. Those rats which had received inescapable shock showed a sharp decline in aggressive behaviors, which persisted even after 96 hours, and a transient increase in defensive behaviors. No change in behavior was seen in those rats which could escape shock or which were not shocked. The experimenters concluded that "learned helplessness" produced a "striking long-term reduction in aggression."		
Maternal Aggression - 1981 Female rats with a newborn litter were mated again to produce a second litter before the first was weaned. The mother rats attacked pups from the first litter, knocking them down, causing them to squeal, and occasionally biting them. Often, the mother would "pin" the pup against the floor with her body. Pups responded to attacks by fleeing or remaining "immobile" in fear for long periods of time.	university	National Science Foundation and National Institutes of Health
Cancer Drug Research - 1982 Previous experiments had shown that hypothyroidism (impaired thyroid function resulting in low levels of thyroid hormones in the blood) produced lesions in dogs similar to those produced by long term administration of a cancer treatment drug, Adriamycin (ADR). Thus, researchers used 20 Beagle dogs to observe the effects of thyroid hormone supplementation on chronic Adriamycin-induced cardiotoxicity (heart poisoning). The dogs were divided into 4 groups (A, B, C, and D). The animals in groups A, B, and C (experimental groups) were given Adriamycin once a week for 20 weeks. Group D was not given ADR and was used as a control group. The dogs in group B and the control group (D) were given daily oral doses of L-thyroxine (a thyroid hormone) at "2 times [the] recommended... dose." The dogs in group C were given "8 times [the] recommended... dose." Group A was not given L-thyroxine. "Dogs that survived the 20-week experimental period," including the controls, were then killed for autopsy. Dogs that died during the experiment were also autopsied. The ADR-treated dogs developed progressive skin discoloration and hair loss after about 4 weeks. Skin discoloration was most prominent over the face and neck, whereas hair loss was seen all over the animals' bodies. An "ulcerative" rash eventually developed on the hairless skin. The animals lost weight, and 5 dogs had "1- to 2-week-long episodes of lethargy and fever." Many dogs had "persistent conjunctivitis" (eye irritation). One dog had "chronic coughing and dyspnea [shortness of breath]" and died with "extensive pneumonia." Five other dogs also died during the experiment, 2 of	veterinary school	American Cancer Society

Description	Type of Institution	Source of Funds
pneumonia, 2 of "congestive heart failure," and 1 of "no established cause." At autopsy, all ADR-treated dogs except 2 were found to have heart damage. The experimenters concluded that "significant effects of [L-thyroxine] supplementation on the frequency or severity of ADR-induced myocardial damage were not seen."		
Deprivation of Sight, Hearing and Smell Reduces Pup-Killing - 1982 Rats were used in two experiments to observe the effects of a chemical called para-chloro-D,L-phenylalanine (PCPA, which depletes the brain chemical serotonin), known to produce a high incidence of pup-killing or mouse-killing "in rodents." In experiment 1, 33 female rats were injected in the neck with PCPA or a glucose solution (controls), then placed in cages with 3 - 7 day old pups to observe the following behaviors: locating (sniffing, nosing and licking the pup), carrying the pup in the mouth, "mauling" (holding the pup down with both feet and biting at it), attack (the first "seizing bite"), "gnawing" (hard, repetitive biting and tearing at the pup with the incisors), and "consumption" (eating the pup). The experiments were repeated once each day for 10 days. The PCPA rats were rated as "strongly filicidal" by the 6th day of testing. Twenty-six additional rats were also injected with either PCPA (20 rats) or glucose solution (6 rats), and these animals were killed at various intervals to measure levels of brain serotonin, which was significantly decreased by PCPA administration. In experiment 2, to observe the effects of "sensory deprivation" on PCPA-induced filicide, 24 female rats were divided into 6 groups and subjected to "sensory impairments" as follows. The rats in the first group were blinded ("visual enucleation") by inserting a syringe needle into the animals' eyes and withdrawing some of the fluid, which was replaced with Zephiran chloride (an antiseptic). The rats in the second group were also blinded and their olfactory bulbs were surgically removed (sense of smell destroyed). The third group was blinded, and subjected to "auditory destruction" (deafening), in which a needle was used to pierce the ear drum and dislodge the middle ear bones. The fourth group was subjected to olfactory bulb removal and "auditory destruction." The fifth group was subjected to all three procedures, and the sixth group was used as a control. After recovery from surgery, the animals were injected with PCPA or a glucose solution and placed in cages with 3 - 7 day old pups as in experiment 1, to observe filicidal behavior. Sensory deprivation greatly reduced the incidence of PCPA-induced filicidal behavior. These experiments demonstrated that pup-killing behavior "seems to be a type of predatory aggression, characterized by three discrete phases: location (including investigation [sniffing, nosing the pup]), mauling, and eventual attack."	university	not acknowledged
Drug Development - 1983 In an 8-month long experiment involving drug self-administration, catheters were implanted into the veins of monkeys, then they were taught to press a lever to receive automatic injections of opiates (heroin or Dilaudid, a morphine prescription drug). A colored light signalled that a lever-press would produce food. Once the animal became addicted to these drugs, the researchers injected other	medical school	National Institute on Drug Abuse

Description	Type of Institution	Source of Funds

drugs, methadone or buprenorphine (currently used to treat human drug addicts). Buprenorphine caused the monkeys to stop lever pressing for the 2 opiates, but had no effect on lever pressing for food. Methadone initially had no effect on lever pressing for opiates, so methadone dose levels were repeatedly increased until one of the monkeys stopped lever pressing for opiates. This monkey died a few days later of an abdominal hemorrhage caused by a ruptured vein. Only 3 monkeys were "able to tolerate" the highest doses of methadone, and many of the surviving animals suffered methadone toxicity, which included seizures, intoxication and decreased breathing rate, accompanied by severely decreased lever pressing for food. In some cases, it was necessary to administer naloxone, an opiate antagonist, to reverse the toxic effects of methadone. "Comparable methadone toxicity" at these doses had been reported by previous researchers.

Animal Model of Tardive Dyskinesia - 1983 — hospital — Veterans Administration

Supported by the U.S.A., researchers from the U.S.A. and Denmark artificially produced an animal model of tardive dyskinesia ("involuntary" movements, usually of the mouth region, often seen in humans after prolonged treatment with behavior-modifying drugs). They injected apomorphine (a morphine derivative which enhances the effects of dopamine, a brain chemical) into monkeys which had previously received daily doses of haloperidol (itself a dyskinesia-inducing drug) for 4-14 months. Apomorphine caused symptoms of tardive dyskinesia in the monkey, including excessive licking and chewing (sometimes of the cage bars or their hands, but often chewing at nothing), protruding their tongues, "grimacing" or gaping their mouths, repetitive movements of the head, neck and trunk, hyperactivity, and increased blinking. The monkeys were allowed to recover, then they were injected in succession with several drugs which enhance the effects of the brain chemical GABA (GAG, muscinol, THIP, picrotoxin) to compare their effects with those of apomorphine. Only one of these drugs produced tardive dyskinesia. However, all drugs produced moderate to severe vomiting and gagging, and one (picrotoxin) produced epileptic seizures and convulsions.

Materials Offgassing - 1982 — private industry — not acknowledged

Mice were used "to assess the possible contribution of dyes to the toxicity of burning textiles" by exposing them to gases from dye-saturated and undyed cottons decomposed at 800°C (1, 472°F). The apparatus consisted of a clear plastic animal exposure chamber, a "furnace," and a pyrolysis tube fitted with a "boat" to hold the decomposing textiles. The tube led into the animal exposure chamber.

The mice were given several minutes to "accustom themselves to their surroundings," then the pyrolysis tube containing the cotton sample was placed into the furnace which was heated either slowly "to stimulate the pre-ignition stage of a fire," or rapidly to 800°C. The heat decomposed the cotton, producing toxic gases which passed into the animal exposure chamber through the tube. The mice were observed for time to incapacitation (staggering, prostration),

Description	Type of Institution	Source of Funds
time to convulsions, time to collapse, and time to death. The test was scheduled to last for 30 minutes "unless 100% mortality occurs earlier." Each experiment was repeated two or more times to provide "measures of variation between test animals and between experiments," and the results were averaged for statistical analysis. Averaging of all experiments showed that after approximately 10 minutes, all mice were incapacitated and convulsed. All were dead within 20 minutes. However, mice exposed to gases from dyed cotton survived longer than mice exposed to gases from undyed cotton.		
Using the same method as described above, mice were exposed to gases from dye-saturated and undyed nylon decomposed at 800°C (1, 472°F). All animals were incapacitated within 20 minutes. Collapse and convulsions occurred by 22 minutes, and all animals were dead by 25 minutes. There was no significant difference in time to death between mice exposed to dyed versus undyed nylon.	private industry	not acknowledged
Memory and Aging - 1981 Young (10 months) and aged (25 months) rats were divided into two groups of both young and old rats. The rats in one group were immobilized in wire cloth tubes. Young rats were then immersed in 5 °C (39.2 °F) water for 12 minutes and old rats immersed in 1 °C (33.8 °F) water for the same time. Both groups of rats were then placed in the white chamber of a black and white shuttlebox. When the rats crossed to the black chamber, they received an inescapable foot shock, which was repeated until the rats learned to avoid footshock by remaining in the white chamber. The purpose was to observe the effects of cold exposure on the rats' ability to learn to avoid electric shock. Then the animals who had not previously been cold-exposed were immobilized in wire cloths and some of these immobilized rats were then immersed in cold water as described above. 24 hours later, they were again placed in the shuttlebox to see whether the cold-exposed and immobilized rats still remembered their previously learned avoidance of the black chamber.	university	University Faculty Research Grant
Alfalfa Seed Poisoning - 1982 Because some people had developed adverse reactions after prolonged eating of alfalfa seeds, a 20-month experiment was conducted in which 5 monkeys (obtained in Indonesia) were fed diets of which 45% was alfalfa seeds. After several months of seed ingestion, 3 of the monkeys "developed signs of a Systemic Lupus Erythrematosus-like illness," a human disease which affects mainly middle-aged women and causes skin eruptions, joint pain, lesions of the internal organs, fever, and many autoimmune reactions in which the body attacks its own cells. The first monkey developed an "inflammatory dermatitis" on the head, arms, and legs which persisted despite antibiotic treatment. Blood sample analysis showed elevated white blood cell count, anemia, immune system alterations, and the presence of autoimmune reactions. The seeds were discontinued after 8 months, but a few weeks later the animal became morose, feverish, anorexic (refused to eat), and the abdomen was distended. "Eleven months after its initial exposure to alfalfa seeds, the animal was moribund and was	primate center	National Institutes of Health and three private foundations

Description	Type of Institution	Source of Funds
killed for autopsy," which revealed widespread skin inflammation, pus- and bacteria-filled skin ulcers, acute peritonitis, and inflammation of the thyroid gland. The second monkey developed "the most remarkable illness during exposure to alfalfa seeds." The animal became lethargic and anorexic after 5 months on alfalfa seeds. Autoimmune reactions and immune system alterations were found in blood samples. After 7 months, a red rash appeared on the animal's face. Alfalfa seeds were withdrawn from the diet, and the monkey was fed a normal diet for the next 10 months. Then, alfalfa seeds were re-introduced into the animal's diet to "further assess the effects of alfalfa seeds." Within 30 days, the animal became lethargic and anorexic. Patches of mange appeared over the face, scalp and trunk, and the monkey's face began to swell. A third of the animal's tail became gangrenous, and the entire tail had to be amputated. Blood sample analysis showed anemia, immune system alterations, and the presence of autoimmune reactions. A renal biopsy was performed, and showed kidney damage. Alfalfa seeds were withdrawn again, since the animal was "considered in danger of succumbing to this illness." The third monkey also became lethargic and anorexic after 6 months on alfalfa seeds, and developed a facial rash. Blood samples showed "persistent anemia," immune system alterations, and autoimmune reactions. Alfalfa seeds were withdrawn after 11 months. The animal was placed on a regular diet for 10 months, then fed alfalfa seeds again. Within 30 days, the monkey became withdrawn and anorexic, and developed a facial rash and patchy mange over the face and scalp. This animal was withdrawn from alfalfa seeds a second time, due to the seriousness of its illness.		
Wound Healing in Esophagus of Ponies — 1983 First, researchers pointed out that complications of surgery of the esophagus can lead to infections, scarring, splitting open of the wound, loss of function, and death. Various suturing techniques have been used, but "The results of those studies indicated that healing may be more dependent on properly placed sutures, gentle handling of the tissue, and using appropriate postoperative care, rather than type of closure used." Then they inflicted esophageal wounds on 12 healthy ponies, using 2 surgical techniques: longitudinal and rotational. These techniques did not influence splitting open of the wounds, but the form of feed given after the operation did. The incisions of all ponies fed mash healed readily, but the wounds of the ponies fed the abrasive hay split open, and by day 8 absesses had formed in 2 of the animals. "The absesses were surgically opened to release saliva and feed" from the tissues. The sutures loosened sooner in the hay-fed ponies than in the mash-fed animals. All ponies were killed 60 days post-surgery.	veterinary school	Biomedical Research Support Grant, National Institutes of Health
Soviet-Style Psychology - 1983 Rats' brains surgically implanted with 3 - 6 electrodes. Following recovery from surgery, the animals were placed on treadmills inside plastic boxes, then electric shocks of varying intensities were delivered to their brains. High shock intensities were aversive, caus-	university	University Project Grant

Description	Type of Institution	Source of Funds
ing the rats to leap up and try to escape the shock. Lower shock intensities caused the rats to walk on the treadmill, alternately flex and extend their forelimbs ("paw flopping"), make digging movements on the treadmill, or turn their heads or bodies. They were then killed and their brains examined to verify placement of the electrodes. The researchers performed their experiments after a search of the Soviet literature.		
Fear of Humans - 1982 Previous experiments had shown that the "mere presence of a human" during "open field testing" prolonged "freezing," as if to "avoid detection" by a predator, in both chickens and rodents. In the present experiments, 4½ week old chicks were used to further observe the effects of the presence of the experimenter during open field testing. In experiment 1, 44 chicks were divided into 2 groups. Each chick from the first group was placed individually in the "open field box", a wooden box with a Plexiglas lid in which ventilation holes had been drilled, while the experimenter sat motionless directly in front of the box, facing the chicks. The chicks in the second group were also placed individually in the open field box; however, the experimenter sat with his back to these chicks. In both groups of chicks, the following were observed: time to distress calls, time to movement, time to attempts to escape from the box by flying or jumping out, and number of defecations. When the experimenter was facing the chicks, they "froze" as if to "avoid detection" for significantly longer periods of time before crying out, moving about the box, or attempting to escape than the chicks who saw only the experimenter's back. Defecation rates were lower in chicks who faced the experimenter. In experiment 2, 27 chicks were divided into 3 groups and placed individually in the open field box as in experiment 1, except that instead of sitting with his back to the second group of chicks, the experimenter covered his face with a sheet of paper, since previous experiments had shown that a stuffed hawk with a hood over its head was not perceived as threatening by chickens. For the third group the experimenter left the room while the chicks were in the open field box. As in the first experiment, the chicks who could see the experimenters face "froze" for longer periods of time before crying out and trying to escape than chicks in the other two groups. There were no significant differences in behavior between the chicks who could not see the experimenter's face and those who could not see the experimenter at all, nor was there any significant difference in defecation rate between all 3 groups. In experiment 3, 60 chicks were divided into 3 groups to observe the effects of distance between chicks and experimenter on "freezing" behavior. These chicks were tested on a white Formica table top instead of in the open field box so that the chicks would be able to see the experimenter at greater distances. Each chick was placed on the table, then the experimenter walked away to a distance of 60 cm (24 inches) from the first group, 200 cm (80 inches) from the second group, and 340 cm (156 inches) from the third group. Those chicks in group 1, which were closer to the experimenter, "froze" for significantly longer periods of time before crying out and trying to	university	National Science Foundation

Description	Type of Institution	Source of Funds
escape than the chicks in groups 2 and 3, which were farther from the experimenter. In experiment 4, 22 chicks were placed individually in the open field box with the Plexiglas lid up. Once the chick had remained quiet for 60 seconds, the experimenter, who sat in front of the box, either reached for the chick (without touching it) or remained quietly seated. Those chicks for whom the experimenter reached ran away, as to avoid a predator, while the chicks who were not reached for remained "frozen" as if to "avoid detection" for long periods of time before crying out and trying to escape. The experimenter concluded that "human contact is aversive [to laboratory animals] and humans are reacted to by laboratory animals in much the same way as they would react to predators under natural conditions."		
Fear of Humans - 1982 "Nervous" pointer dogs, specially bred to be fearful of humans in a program which began in 1969, were injected with pimozide or chlordiazepoxide (2 anti-anxiety drugs) to observe the behavioral effects of these drugs. The "nervousness" of the dogs was assessed both before and after drug administration by observing frequency of such behaviors as retreating, "posturing" in a catatonic position (remaining in abnormal position for several minutes), circling, trembling, urinating, and defecating.	government - non-military	Veterans Administration
Fear of Humans - 1983 "Nervous" pointer dogs have been specially bred to fear humans in a program which began in 1969. These dogs, which become catatonic and trembling in the presence of humans, were injected with pimoxide, chlordiazepoxide, or naloxone to observe the effects of these drugs on muscle rigidity and heart rate of the animals. Neck muscle rigidity, measured by the force needed to pull the blindfolded dog's head to the side, was reduced significantly by all three drugs. Heart rate, as recorded by needle electrodes inserted in the right shoulder and left hip, was increased by naloxone, but was unaffected by pimozide or chlordiazepoxide.	university	Veterans Administration
Fear-Motivated Behavior - 1983 To observe the effects of "psychological stress" on prolactin (a hormone) release, a cannula was implanted into rats' veins to obtain blood samples, and the animals placed in "novel environments to generate fear motivated behavior." These included handling, "sitting" the rats in water, placing the animals in an unfamiliar cage, or on a platform over water. All procedures resulted in increased prolatin levels in the blood. As the rats "habituated" (became used to) the novel environments, their fear subsided and prolactin levels returned to normal.	university	not acknowledged
Yellow Fever - 1983 After citing references on research back to 1930, 10 rhesus monkeys were injected with yellow fever. "When death appeared imminent," the monkeys were killed to observe biochemical changes.	government - military	Army

Description	Type of Institution	Source of Funds
Animal Model of Thiamine Deficiency - 1983 Monkeys were used to artifically produce an animal model of Wernicke-Korsakoff Syndrome, a disease caused by thiamine (Vitamin B_1) deficiency known to produce loss of memory, disorientation, and brain damage in alcoholics. Seventeen monkeys were divided into 5 groups and subjected to one or more "bouts" of thiamine deficiency by feeding the animals a diet deficient in thiamine until "characteristic symptoms" appeared, including prolonged anorexia (refusal to eat), vomiting, and inactivity. When thiamine deficiency became "severely life-threatening" the animals were given thiamine injections to "induce reversal of symptoms," then the animals were fed a normal diet and allowed to recover. The second group was also subjected to one "bout" of thiamine deficiency; however, after recovery from this "bout" the animals were subjected to a second "bout" of thiamine deficiency. The third group was subjected to 4 "bouts" of thiamine deficiency in the same way. The fourth group, a "pair-fed" control (PFC) group, received the thiamine-deficient diet but was given thiamine supplements. The last group was fed normal monkey chow and used as controls. The monkeys were examined during the "bouts" of thiamine deficiency for possible signs of brain and nerve damage, including abnormal muscle tone and reflexes, staggering during a "simple walking test," mouth sag, facial contractions or stiffness, "tremor" of the tongue or limbs, "rebound phenomenon" (inability to stop movement caused when the experimenter suddenly released a restraining hold on the animals forearms), withdrawal from cotton touched to the skin or from pinpricks, abnormal eye movements, and vision disturbances. All monkeys, including the pair-fed and normal controls, were killed following the experiment to observe location of brain damage. In the first group, initial symptoms of thiamine deficiency were "anorexia, adipsia [refusal to drink], and weight loss." Symptoms of the next stage of thiamine deficiency included decreased activity, and "prolonged squatting" in which the animal sat "hunched in one corner of its cage with its head slightly bowed between its legs." The animals became progressively weaker, expecially in the legs, staggering when forced to walk, began to vomit, and finally collapsed. Signs of nerve damage included visual abnormalities, dilated pupils, nystagmus (eye rolling), drooping eyelids, and abnormal tendon reflexes. Tremors, limb incoordination, and "rebound phenomenon" did not occur. This group was then given thiamine injections to reverse the symptoms. One monkey had become "so weak and dehydrated" that thiamine injections were "insufficient to induce recovery" so that fluids had to be administered intravenously and the monkey had to be " force-fed." This animal finally recovered from thiamine deficiency; however, "weakness in the lower extremities persisted for several weeks, and full strength of the leg muscles was never regained." Monkeys in the second group showed symptoms of thiamine deficiency similar to the first group. These symptoms were also seen during the second "bout" of thiamine deficiency, but a shorter period of deficiency was required "for most serious symptoms to develop." All animals recovered within 24 hours after thiamine administra-	government - non-military	National Institute of Mental Health and U.S. Public Health Service

Description	Type of Institution	Source of Funds
tion. The animals in the third group showed these same symptoms "throughout the four periods of deprivation" and followed the same course of recovery as the first and second groups. Following the final recovery period, all monkeys including controls were subjected to several behavioral tests, in which they were rewarded with food for performing a variety of tasks involving memory. The monkeys who had been thiamine deprived were not able to perform the tasks as well as the controls, indicating memory loss resulting from thiamine deprivation. At necropsy (autopsy), examination of the brains of these monkeys revealed lesions in various brain areas. Damage was more severe and affected a greater number of brains areas in monkeys which had more than one "bout" of thiamine deficiency.		
Animal Model of Hemorrhagic Shock - 1983 To artificially produce a "conscious" animal model of hemorragic shock, 25 pigs were implanted with a silicon rubber catheter in either the carotid artery (in the neck) or the descending aorta (heart artery). After 5 days the animals were bled. Removal of 55% to total "estimated" blood volume from the carotid artery over 66 minutes led to 86% of deaths within 73 minutes. Removal of 60% of total "estimated" blood volume from the descending aorta over 60 minutes produced no deaths; however, 33% deaths resulted from removal of the same amount of blood in 28 minutes, and 100% deaths occurred when the blood was removed in only 14 minutes. The experimenters concluded that there is a "markedly different mortality rate between carotid artery and descending aorta catheter placements...and the pig is very resistant to hemorragic shock mortality."	government - military	Army
Constipation Deaths - 1982 Veterinary researchers pointed out that mucoid enteritis is a common disease of young rabbits, causing loss of appetite, inability to drink liquids, diarrhea, dehydration, low rectal temperature, loss of 20-25% of body weight in 1-2 days, weakness, crouched posture, roughened coat and bloated abdomen, and the disease usually results in death. Thus, they "attempted to reproduce" the disease in healthy rabbits by tying off various segments of the gastrointestinal tract, to simulate constipation or impaction (known to produce mucoid enteritis in normal rabbits). In 11 rabbits, the colon was tied off (ligated) tightly enough to prevent passage of the intestinal contents. In 40 other rabbits, the cecum (the beginning of the colon) was ligated; however, this did not affect the flow of solid waste material to the rectum. Part of the cecum was removed in 5 additional rabbits. All 11 rabbits with ligated colons were unable to eat or drink after 2 days and died within 1 week. These rabbits were unable to pass feces after 2 days and 5 of them began to pass substantial amounts of mucus. "28 of the 40 rabbits with ligated ceca developed clinical signs of mucoid enteritis and died within 8 days." These rabbits were mildly dehydrated, unable to eat or drink, showed weight loss, and passed substantial amounts of mucus. All rabbits with part of the cecum removed died within 3 days of a ruptured appendix or	government - non-military	National Institutes of Health

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peritonitis. Those rabbits which did not become ill were killed after 3 months. At autopsy, none of the rabbits with artificially-produced mucoid enteritis were found to have the bloated stomach and severe dehydration seen in naturally-occuring mucoid enteritis.

Effects of Starvation Plus Trauma - 1981 — university — National Institutes of Health and Army

Rats were starved for either 24 hours or 5½ days, then the animals' ceca (part of the small colon) was surgically exposed and ligated (tied off) to observe the effects of starvation and trauma (cecal ligation) on survival, liver metabolism, and the reticuloendothelial system (responsible for removing foreign matter from the blood stream). Other rats, also starved for 24 hours or 5½ days, were "sham-operated" (anesthetized and abdomen surgically opened but no cecal ligation performed). Operated animals were considered to have "survived" if they "lived at least 5 days" following surgery. A third group of rats was starved for 7 or 11 days to observe the effects of long-term starvation only.

In intact rats, starvation of 7 days resulted in 32% weight loss, but no deaths. Rats starved for 11 days lost 44% of body weight and all died of starvation. In the operated rats, no deaths were seen in either cecal-ligated or sham-operated rats starved for 24 hours. 60% of cecal-ligated and 20% of sham-operated rats starved for 5½ days before surgery died. Cecal-ligated rats died of sepsis (infection). Alterations were seen in liver metabolism and reticuloendothelial system function in rats with ligated ceca. The experimenters concluded that "prolonged starvation made the animals more susceptible to trauma and infection."

Infant Animals Cry for Food - 1983 — university — National Institutes of Mental Health

Previous experiments had shown that a behavior, such as lever pressing or maze running to obtain a reward (operant conditioning) is more resistant to extinction (the behavior ceases if there is no longer any reward) when the behavior was only rewarded intermittently (50% of the time) than when it was rewarded continuously (100% of the time). This is called the partial-reinforcement extinction effect (PREE) and has been seen in rats as young as 12-14 days old. The present experiment was performed to observe whether the PREE could be seen in 4-5 days old guinea pigs, which mature faster than rats.

Sixteen 4-5 day old guinea pigs were starved and water deprived for 24 hours, then divided into two groups and forced to run down an alley to obtain pureed lettuce. The animals in the first group received lettuce each time they ran the alley, while the second group received lettuce only 50% of the time. The 50% animals initially squeaked "repeatedly" when there was no lettuce at the end of the alley, but after several experimental sessions both groups learned to run quickly down the alley. Then "extinction" was begun, in which the lettuce was no longer placed at the end of the alley for either group. The continuously-reinforced (100%) animals in group 1 vocalized "vigorously" when there was no lettuce at the end of the alley, and they ran progressively slower or stopped running altogether (behavior extinguished). The partially-reinforced (50%) animals in

Description	Type of Institution	Source of Funds
group 2 were resistant to extinction and continued to run quickly down the alley (PREE). The experimenters concluded that PREE can be observed in guinea pigs as young as 4-5 days old.		
Poisoning of Ponies with PBZ (Bute) - 1981 In 1981, Scottish veterinarians published in an American veterinary journal an account of the effects of phenylbutazone (PBZ), that familiar anti-inflammatory, analgesic drug widely known as Bute, commonly used to keep lame horses racing. The veterinarians stated that "until recently," Bute was considered non-toxic to horses. However, they noted that fatalities could be induced in horses using the "recommended" doses of 8-14 milligrams of Bute per kilogram of body weight (doses much higher than the "recommended" doses cited by other experimenters), and "suggested" that ulceration and increased water retention caused the toxic effects. Thus, to "study further" the causes of Bute poisoning in horses, they performed 3 experiments. In the first experiment, they administered high "recommended" doses of PBZ to 5 ponies by adding it to their feed for 8 days (3 ponies) or 10 days (2 ponies). After test day (TD) 4, pony 1 was given a "bolus" containing PBZ because it would not eat the Bute-spiked feed. By TD 6, this pony was depressed and spent long periods lying down. Its oral tissues turned blue, its extremities were cold, and its rectal temperature and heart rate were abnormal. The "treatment" (poisoning) was continued, and "on TD 8, the pony died." It had lost 10% of its body weight "during treatment." By TD 8, pony 2 was recumbent for long periods and was euthanized. It had lost 13% of its weight. Pony 3 appeared normal, but was killed on TD 8. Pony 4 remained normal for 8 days, but by TDs 9 and 10, it would no longer eat its drugged feed, and "died suddenly during the night of TD 10." It had lost 6% of its body weight. Pony 5 remained normal but was also killed. Necropsies (autopsies) were performed on all animals. In ponies 1 and 4, the "primary finding was massive intestinal ulceration" in the large colon and cecum, with a few ulcers in the duodenum and stomach of pony 1. The cecum and colon were "peppered" with numerous "erosions." Microscopically, there were various necrotic (rotting) areas, with large masses of bacteria evident. A similar range of gross and microscopic lesions were found in ponies 2, 3 and 5, but there were variations in the degree of severity. "In pony 2, ulcers in cecum and colon were similar to those found in the ponies that died; in addition, there were ulcers on the lateral side of the tongue of pony 2." Pony 3 had a large number of erosions in the cesum and colon, but no frank ulceration, whereas pony 5 had only one ulcer on the side of the tongue. In experiment 2, moderate "recommended" doses of PBZ were administered to 4 ponies (ponies 6, 7, 8 and 9) in their feed for 6 days, and 2 ponies (ponies 10 and 11) were used as controls, All 6 ponies received daily injections of radiolabelled chromic chloride to observe protein loss from the gastrointestinal tract, as measured by radioactivity in blood samples and feces. Ponies 6 and 7 remained normal during PBZ treatment, and were euthanized and autopsied on post-treatment day 1. Pony 8 became quiet and depressed on the last day of treatment, but returned to normal with a few days. On TD 3, pony	veterinary school	Horserace Betting Levy Board

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9 was quiet and on TD 4 refused to eat the PBZ in the food; therefore, a "bolus" of the drug was given. On TD 5, oral ulceration was observed, the pony was depressed, and ate and drank little. The animal appeared to recover, but on posttreatment day 42, the condition of pony 9 deteriorated, it became recumbent and depressed, and ate only moderately. Although slight improvement was observed several days later, the pony remained depressed and was killed on posttreatment day 53. A "marked loss in body condition was observed" and muscle atrophy was seen all over the body. Pony 9 was the only animal in experiment 2 that had a weight loss (14%) by TD 6. Although a high-protein diet had been offered the animal posttreatment, the pony continued to lose weight. By posttreatment day 29 the pony weighed only 72% of its pretreatment weight, and on posttreatment day 53 it weighed only 66% of its pretreatment weight. Greatly increased radioactivity was seen in the blood and feces of the PBZ treated ponies, indicating loss of radiolabelled protein from the gastrointestinal tract. "To investigate the site of gastrointestinal loss," ponies 6 and 7 were euthanized and radioactivity of the gastrointestinal contents was measured. Highest levels of radioactivity were seen in the stomach contents of pony 6 and the small colon contents of pony 7, indicating that the highest loss of protein occurred in these areas. Autopsy revealed that peritonitis had developed in pony 9. Experiment 3 involved the metabolism of PBZ using lower doses. No fatalities, abnormalities, or euthanasia were reported.		
Poisoning of Ponies with PBZ (Bute) - 1983 Following publication of the above experiments in a widely-read American veterinary journal, American veterinarians said that "there have been few reports of phenylbutazone [Bute] toxicity in horses," and performed experiments to see if Bute poisoning could be reproduced "in the pony." They cited "recommended" doses of Bute far lower than those reported in the above experiments, although they used doses of Bute which were consistently higher than the "recommended" dose they cited. In Study 1, 7 ponies were administered an initial intravenous dose of Bute, followed by 13 daily oral doses. The hair was clipped from their abdomens and a hole was made in the abdomen for the collection of peritoneal fluid. In Study 2, 3 ponies were administered Bute orally for 14 days, and 3 ponies were used as controls. Six of the ponies given Bute "died during the study-the earliest at 7 days and the last at 20 days." Another pony was euthanized on the 10th day, "during an acute abdominal crisis." Still another pony "recovered from the initial study and was found dead a few weeks later," with a ruptured colon. The first signs of poisoning occurred on the 5th to 8th day postdosing. The animals became lethargic and depressed, and they stood with lowered heads. 7 ponies developed "oral ulcers." By the 8th to 11th days, most ponies refused to eat grain, but continued to eat small amounts of hay. "Fluidy feces (cow-like) occurred in 6 ponies. Three of these progressed to profuse diarrhea before death occurred." Necropsy (autopsy) of the drugged ponies revealed oral and	veterinary school	not acknowledged

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gastrointestinal ulcers, colitis, peritonitis, and necrosis (rotting) of some kidney structures. Peritoneal fluid samples from the ponies in Study 1 showed drastically increased white blood cell count. Seven of the ponies given Bute showed weight loss of up to 25 kg (55 lb) after 10 days.		
Rotation-Induced Vomiting - 1980 To artificially produce an animal model of motion sickness, a "study of susceptibility" to motion sickness was performed using 104 Bolivian and Columbian squirrel monkeys of both sexes. The monkeys were placed in a "rotation cage," and spun counterclockwise at speeds of 5, 25, and 50 rpm, together with a "sinusoidal" (wavelike) up and down acceleration, for 60 minute periods to observe time-to-vomiting, frequency of vomiting, and occurrence of the "Sopite Syndrome" in which the rotated monkeys appeared drowsy and assumed a "crouching defensive body posture" with the head placed between the legs, the hands grasping the head and the tail curled over the shoulders, even when they did not vomit. A blackened tube was placed around the Lucite rotation cages of some monkeys so they could not see while being spun, while other monkeys were allowed to watch the spinning environment. Conclusion: (1) high rotation speeds produce more vomiting than low rotation speeds, (2) Bolivian monkeys are more susceptible to motion sickness than Columbian monkeys, (3) males are more susceptible than females, (4) monkeys which could see the spinning environment vomited more than monkeys which could not, and (5) appearance of the "Sopite Syndrome" indicated motion sickness, even in the absence of vomiting.	primate center	National Institutes of Health, and National Aeronautics and Space Administration
Motion Sickness - 1980 Squirrel monkeys were placed in "translucent plastic cages," fitted with circular horizontal rotating platforms and spun counterclockwise at 25 rpm, together with a "sinusoidal" (wavelike) up and down acceleration of 6 inches every 2 seconds. The animals were spun for 60 minutes once each week to observe time-to-vomiting and frequency of vomiting. The "ultimate criterion" of vomiting was "forcible expulsion of the stomach contents through the oral fissure [mouth]." All monkeys vomited within approximately 20 minutes, and some vomited more than once. After the third week, an area of the brain (area postrema) of 8 animals was electrically ablated. Two of the monkeys were sham-operated (anesthetized and their skulls surgically opened but no brain ablation performed), and 2 monkeys were not operated on. These 4 monkeys served as controls. All of the animals were then spun again. This time, some of the monkeys who had been brain ablated did not vomit. The brain-ablated monkeys were then killed to verify the area ablated. Both the sham-operated and non-operated controls were also killed. These experiments "appear" to agree with similar experiments performed in 1952 and 1954.	government - non-military	National Aeronautics and Space Administration and National Institutes of Health
Radiation Sickness - 1980 Chair-restrained monkeys were exposed to various doses of ^{60}Co (cobalt-60) radiation to observe time-to-vomiting and frequency of vomiting and retching. Retching was often accompanied by	government - military	Air Force

Description	Type of Institution	Source of Funds
vomiting; however, monkeys who vomited often held their vomitus in their cheek pouches and sometimes swallowed it. All monkeys, whether vomiting or not, showed signs of "malaise," including grimacing and facial contortions. One monkey who did not vomit showed signs of severe nausea, including "vigorous 'mouthing,' grimacing, some coughing, and some retching, but not enough to be considered a nonproductive emetic episode." Another monkey vomited 14 times. A second group of chair-restrained monkeys were pivoted forward and backward on a moving platform while being irradiated, to observe the combined effects of motion and radiation on time-to-vomiting and frequency of vomiting and retching. Vomiting, retching, and nausea were produced at lower radiation doses in these animals than in those which remained stationary during irradiation.		
Behavioral Changes in Irradiated Dogs - 1983 Twelve beagle dogs were used to observe the effects of radiation to the head on behavior. Prior to irradiation, the dogs were videotaped for 24 hours, to observe the time spent jumping on the cage walls, moving about the cage, standing, sitting, and lying down. The dogs were also taught to run through a maze, to observe the number of wrong turns (errors) the animals made before finding their way out. Six of the dogs were then anesthetized, placed in a sling, and exposed to either 1000 (low dose) or 1750 (high dose) rads of Cobalt-60 (^{60}Co) radiation to the head. The remaining dogs were "sham-irradiated" (anesthetized and placed in the sling but not irradiated) and were used as controls. Following irradiation, one dog was "hospitalized for three days of intestinal upset with accompanying bloody and mucus-filled diarrhea." Two dogs which had received low dose radiation developed "occasional head shaking." Two other dogs which received high dose radiation developed head shaking so severe that their ears became "irritated and infected." All dogs were again videotaped following radiation exposure. Those dogs which received high doses of radiation spent more time sitting and lying, and less time jumping and moving. When placed in the maze, the irradiated dogs made significantly more errors than the controls. One dog which received high dose radiation was "extremely fearful of events associated with running the maze." CT scans (brain x-rays) showed no brain lesions in the irradiated dogs, even though the experimenters had expected to find some. They concluded that "behavioral changes can and do occur" in the absence of brain damage.	veterinary school	National Aeronautics and Space Administration
Penile Function in Mutilated and Drugged Rats - 1983 Male "sexually experienced" rats were used in 3 experiments to observe the effects of RDS-127, a dopamine (brain chemical) agonist, on penile reflexes and seminal emission (ejaculation without intromission). In preliminary experiments, the rats were rated on their "performance in copulatory tests" by observation of mounting frequency, time-to-intromission after a female was placed in the cage, number of intromissions before ejaculation, and length of time between ejaculation and next intromission. In experiment 1, 24 rats were injected with either the drug or saline. After 30 minutes "penile	university	National Institute of Mental Health

reflex testing" was begun. The animals were placed on their backs and immobilized in a plastic cylinder with the "penile sheath retracted by a wooden applicator," a procedure used to stimulate penile reflexes. Penile responses were recorded as follows: erections, seminal emissions, "cups" (flaring of the engorged glans penis), "quick flips" (rapid movement of the penis toward the abdomen), and "long flips" (slower flexion of the penis). RDS-127 caused suppression of penile responses, since only 1 out of 12 drugged rats as opposed to 7 out of 12 control (saline) rats showed penile reflexes. In addition, the drug increased incidence of seminal emission, since 5 of the drugged rats had at least 2. The hindlimbs and toes of the drugged rats were "quite extended...and their feet were purple in color." Their penes also had a purplish color "which did not appear to involve engorgement." The drugged rats "struggled more than controls, attempting to crawl [further] into the test cylinder." One rat died within 2 hours of injection.

In experiment 2, the 12 control rats from experiment 1 and 14 additional males were fitted with "thoracic corsets...to prevent the males from bending to groom their genitalia." The penile sheath was retracted and any seminal "plug" material present was removed. The animals were then injected with either RDS-127 or saline, and placed in individual wire-bottom cages, and "closely observed" for appearance of seminal material. The researchers found that 11 of the drugged rats but none of the saline controls produced at least 1 seminal plug within 15 minutes of injection. Production of larger plugs was preceded by "obvious penile activity within the sheath and retraction of the testicles." Three of the rats attempted to groom while plugs were being emitted, but were unable to because of the corset. "Erection in the absence of seminal emission was not observed." The "penis, cage, and papers placed under the cage were [then] closely inspected for previously overlooked seminal material." This inspection was repeated 2 hours after injection, then the test for "spontaneous seminal emission," also referred to as the "corset" test, was begun in which the corsetted animals were left in the individual wire bottom cages for 3 days. The animals' penes, cages, and collecting papers were examined every 24 hours for seminal emissions. At the end of 3 days, there was no difference in seminal emission between drugged and control animals.

In experiment 3, 7 spine-transected animals and 12 "sham-transected" controls were injected with distilled water and immobilized in plastic cylinders. After 15 minutes of observation, the penile sheath was retracted and penile reflexes observed. The spine-transected rats showed erections sooner, and had significantly more "quick flips," "long flips," and "cups" than the sham-transected rats. Two days later, the animals were again immobilized, injected with RDS-127, and testing for penile reflexes was repeated. After injection of RDS-127, penile reflexes were suppressed in spine-transected rats, but all rats had increased seminal emissions. RDS-127 injection had the following effect in the spinal rats: the tail became "very rigid" and moved slowly from side to side. The testes were retracted, and the penes and feet had a purplish color. Hindlimbs and toes were rigidly extended. When released, the animals tried to pull themselves back into the testing cylinder. Both

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the cylinders and the rats were very damp at the end of the experiment due to sweating and salivating of the animals. When placed on a flat surface, the paralyzed hind feet showed "paddling" movements as the animal pulled itself forward with its front feet. One animal died within 2 hours of RDS-127 injection and a second rat died within 12 hours.		
Sexual Repsonses Disrupted by Spine Transection - 1983 Twenty-six ovariectomized(ovaries removed) hamsters were given injections of estrogen and progesterone (female sex hormones) and exposed to a "sexually-active male" to observe lordosis (raising the hind quarters in response to sexual stimulation). "Intromissions by the male were not permitted."' After 2 minutes, the male was removed, and the females' lordosis response to tactile stimulation by the researchers with a small camel's hair brush on the back, flanks, rump, genitals, thorax, and shoulders was observed. During these tests, the male was "reintroduced for brief periods. . . to maintain the highest degree of responsiveness in the female." Then, various "pathways" (groups of nerves) in the spinal cords of the female hamsters were transected (severed) at either the cervical (neck) or chest level of the spine to observe the effects on lordosis. Two additional hamsters were "sham-transected" (spinal cord surgically exposed but not severed). Of the 26 operated animals, 18 "recovered without gross postural or locomotor impairment." All of these recovered animals were then tested for lordosis in response to a male and to stimulation by the researchers with a brush as above. If the spine-severed animal showed no lordosis response to the brush, manual stimulation of the flanks and genitals with the researcher's fingers was applied to produce "pressure in addition to tactile stimulation." In addition, males were repeatedly placed on the backs of unresponsive animals "in further attempts to elicit lordosis." Lordosis response was either sharply decreased or totally absent following spine transection as compared to lordosis response without spine transection or in sham-transected hamsters. All animals, including sham-transected controls, were killed following the experiment to verify spinal cord area damaged.	university	National Institutes of Health
Sexual Responses in Spine-Transected Rats - 1981 13 male, spine-transected rats were used in two experiments to observe ejaculation and penile reflexes elicited by electric shock to the legs. All spine severed rats received daily injections of testosterone (male sex hormone) to avoid any changes in penile reflexes due to "possible testicular atrophy after transection." Preliminary tests were conducted, in which the rats were immobilized in plastic cylinders and the penile sheath retracted for 15 minutes. "All subjects displayed the expected erections and flips occuring in clusters every 2-3 minutes." During experiment 1, the rats were again immobilized and given electric shocks every 2 minutes for 15 minutes through "wound clips placed bilaterally [one in each leg] into the skin of the flank region." The penile sheath was not retracted. This procedure was repeated from 1 to 26 times, at 2-3 day intervals, depending on the animal's responses. Electric shock was followed by "twitching of the spinal column and back legs." About 20 seconds	veterinary school	National Institute of Mental Health

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later, ejaculation occurred, "preceded usually by two or three erections, . . . a train of three to four flips followed the onset of ejaculation and continued while the seminal material was coagulating." In experiment 2, 4 of the rats that "responded very consistently" in experiment 1 were subjected to each of 3 procedures. The first was identical to the procedure in experiment 1: the penile sheath was not retracted and electric shocks were delivered to each flank every 2 minutes. The second procedure was similar to the first, except that the penile sheath was retracted. The third procedure was also similar to the first, except that the electric shocks were both given to the same leg instead of one shock to each leg. The first procedure in experiment 2 produced erections and ejaculations similar to those previously seen in experiment 1. The second procedure resulted in erections and penile reflexes, but only 1 rat ejaculated. The third procedure produced no penile responses whatsoever.		
Anesthetized Penis Causes Sexual Impotence - 1983 Male rats were used in two experiments to observe "penile reflexes" in the anesthetized penis. In preliminary experiments, the rats were immobilized in plastic cylinders and the penile sheath retracted to observe expected penile reflexes, including erections, "cups," "quick flips," and "long flips." Only rats which showed penile reflexes within 15 minutes were used. In experiment 1, 9 rats were repeatedly tested for penile reflexes following applications of pontocaine (a topical anesthetic) or saline. While penile reflexes were seen in all animals following saline applications, only one animal showed reflexes after anesthetization. This rat began to display penile responses and ejaculation while the anesthetic was being applied, and these continued throughout the entire test. The animal was later retested with pontocaine, and again showed the same "unusual response." In experiment 2, 12 rats were placed with females and allowed to ejaculate during copulation, then they were immobilized, their penes swabbed twice with saline, and the penile sheath retracted. Penile reflexes were observed much sooner in these rats than in rats swabbed with saline in experiment 1. The procedure was then repeated, using pontocaine instead of saline. Anesthetization of the penis did not prevent penile reflexes in these rats. The animals were then returned to the "mating arena" and "permitted to attempt copulation"; however, they were unable to achieve intromission despite "repeated attempts."	university	National Institutes of Health
Sex in Ovariectomized and Brain-Ablated Cats - 1982 Seven "mongrel" female cats, whose "prior reproductive history was unknown," were used to observe the effects of brain ablation on estrous ("heat") in ovariectomized (both ovaries removed) animals. Following surgery, the cats were given injections of replacement estrogen (female sex hormone). Preliminary tests were conducted to observe whether the cats were "typical of the behavioral norm" seen in previous experiments, which showed that injections of replacement estrogen would produce full estrous behavior in the operated cats, and withdrawal of replacement estrogen would result in total disappearance of the "heat" responses. "Estrous behavior" was observed by placing each cat in a "cubicle," and probing the vagina	university	National Institutes of Health

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"manually" with an "oil-lubricated, 6mm-diameter blunt Teflon probe" which was inserted into the animal's vagina "until resistance was felt." Upon completion of preliminary tests, the central gray areas of the animals' brain were electrically ablated in 5 cats. The remaining 2 cats received "control" ablations in other areas of the brain. "Estrous behavior testing" by vaginal probing was resumed in all cats on the second day after brain ablation and continued for 26 to 61 days, both with and without replacement estrogen injections. Behavioral responses to vaginal probing in cats, both brain-ablated and non-ablated, which received replacement estrogen were as follows: lordosis (raising of the hind quarters), tail deflection, treading of the hind feet, "estrous cry," and "after-reaction" (licking of the genitals, vigorous rubbing and rolling). Non-brain-ablated cats which did not receive replacement estrogen showed "hostility and resistance" to vaginal probing, and tucked their tails tightly around their genital region. Ablation of the central gray brain area produced "persistent estrous behavioral responses [even] in the absence of estrogen replacement" and caused "complete and permanent abolition" of hostility and resistance to vaginal probing seen in non-brain-ablated cats. No changes were observed in cats with "control lesions" in other brain areas. Following experimentation, all cats were killed and their brains examined to verify area ablated.		
Stress-Induced Ulcers - 1982 173 rats were used in 3 experiments to observe whether increased incidence of stomach lesions (ulcers) previously seen in rats allowed a "rest period" following electric shock stress could be repeated using immobilization stress, and to artificially produce an animal model of ulcers. In experiment 1, 60 rats were food deprived for 24 hours, then immobilized on their backs, held securely on the "restraint boards" by leather flaps drawn over their thorax and abdomen. The legs were "drawn out from the body at a 45° angle and secured with leather loops." The immobilized rats were then placed in a "refrigeration chamber" at a temperature of 4°C (39.2°F). After 3 hours, the rats were removed from the chamber and released from the restraint boards. Some rats were killed immediately, and the remaining rats killed at intervals of 0.5, 1.0, 1.5, 2.0, or 3.0 hours. All rats had stomach lesions; however, incidence of ulcers was significantly greater in those rats killed after 1.5 hours of "post-stress rest" than in rats killed immediately after stress. In experiment 2, 63 rats were food deprived and immobilized for 3 hours as in experiment 1, and some rats were killed immediately after immobilization stress. The remaining rats were killed at intervals of 3, 6, 12, 24, 36, or 48 hours, to observe the effects of long term "post-stress rest periods" on immobilization induced ulcers. All rats developed stomach lesions which were still present after 48 hours. However, a decline in the number and size of the ulcers was observed in rats who had "rest periods" greater than 12 hours. The experimenters concluded that immobilization stress-induced ulcers were a "useful model for evaluating anti-ulcer drugs" since these ulcers do not heal as rapidly as those produced by other "animal models of stress ulcer."	government - non-military	Veterans Administration

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To test this conclusion, 50 rats in experiment 3 were food deprived for 12 hours (shown in previous experiments to increase the "potency" of immobilization induced ulcers as opposed to 24 hours food deprivation), and then immobilized for 3 hours as in experiment 1. One group of rats was killed immediately after immobilization stress, and the remaining rats were injected with either cimetidine (a well-known anti-ulcer drug) or saline for 48 or 96 hours, following which all animals were killed. All rats had stomach ulcers; however, cimetidine-treated rats had fewer and smaller ulcers than saline-treated rats.		
Stress - 1983 First, rats were starved to 80% of normal weight, then were placed in a T-maze with a shock grid floor. They were then foot shocked with low (barely detectable) and high (painful) intensities. Low intensity shock signalled that food was available in one arm of the maze, while high intensity shock signalled that food was available in the other arm. The animals were presented with approximately 3,600 electric shocks over 30-50 sessions until they learned to discriminate between low and high intensity shocks by running into the correct arm of the maze to obtain food. They were then subjected to immobilization stress in Plexiglas restraining tubes for either 30 or 70 minutes, then placed in the T-maze again observe the effects of immobilization stress on shock discrimination. Some of the rats were then immobilized 30 minutes a day for 22 days. Following daily immobilization, they were again placed in the T-maze and electric shocked, to observe the effects of long term immobilization stress on shock discrimination. One rat died of "unknown causes" at the end of the experiment.	university	National Institute on Drug Abuse
Coping with Stress - 1983 To observe the effects of "coping" with stress (electric shock) on the suppression of white blood cell production (associated with cancer growth), rats were placed in a "wheel-turn" box and given one electric shock to the tail per minute for 80 minutes. One group of rats could escape from electric shocks by turning the wheel. A second group of rats could not escape the shocks. 24 hours later, all rats were given 5 footshocks, then were anesthetized and blood samples were taken by heart puncture. White blood cell production was suppressed in those rats which received inescapable shocks, but not in those which could escape from shock.	university	National Science Foundation
Stress-Induced Reduction of Tumor Rejection - 1982 Rats with growing tumors were killed and the tumor cells injected into the left front flanks of 93 healthy rats. 24 hours later, one group of the rats was given 60 electric shocks at random intervals from which they could escape by pressing a lever. A second group of rats was "yoked" to the first group to receive simultaneous but inescapable shocks. A third group of rats was not shocked. 30 days later, all rats were killed to observe the effects of inescapable electric shock (stress) on tumor rejection. Only 27% of the rats who received inescapable shock had rejected the tumor, while 60% of the rats given shocks from which they could escape and 54% of the rats who received no shocks rejected the tumor.	university	not acknowledged

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Endotoxin and Heat Stress Deaths - 1983 Tolerance to endotoxin (bacteria which causes septic shock) was induced in rats by injecting them with progressively increasing sublethal (not enough to kill) doses of endotoxin. Other rats were made overly-susceptible to endotoxin by injections of zymosan. All rats were then challenged by injections of lethal doses of endotoxin to observe decreased number of deaths in tolerant rats and increased number of deaths in sensitive rats. Surviving rats were starved for 18-24 hours, and temperature sensors were inserted into their rectums. They were then subjected to heat stress (41°C or 106°F) for varying periods of time to observe the effects of endotoxin tolerance or sensitivity on heat stress mortality. Endotoxin tolerance decreased number of deaths from heat stress while endotoxin sensitivity increased the number of deaths.	government - military	Army
Urine-Spreading in Heat Stressed Rats - 1981 Citing experiments back to 1947 which demonstrated the importance of salivation and fur-licking to survival in heat-stressed rats, experimenters first desalivated rats (cut out their salivary glands) or injected intact animals with atropine (known to inhibit salivation). Other rats, which had been "sham-desalivated" or injected with saline were used as controls. Some of these rats were also immobilized in "restraint cages." Then all animals were subjected to a temperature of 41.5°C (106.7°F) to observe the time necessary to raise the rats' body temperature to 42.6°C (108.7°F). Desalivation, atropine injection, and immobilization significantly reduced the time necessary to raise the animals' body temperature. The desalivated rats used "urine spreading behavior" in which the heat stressed animals exhibited "voluntary urination" and then rubbed their fur in the urine. Other desalivated rats which were also immobilized could not engage in "urine spreading" and the time necessary to raise their body temperature was further reduced. Atropine injected rats did not use "urine spreading." "A lack of urine spreading activity was probably due to an "inhibition of miction" (urination).	government - military	Army
Heat Stroke Fatalities - 1983 Rats were used in an experiment to observe the effects of previous "endurance training" on the tolerance to treadmill running during heat exposure. The rats were divided into 2 groups: sedentary and trained. The sedentary group remained in their cages, while the trained group underwent a "progressive endurance training program" in which the rats were forced to run on a motorized treadmill 5 days a week for 6 weeks. The speed of the treadmill and the amount of time spent running each day were gradually increased, until during the 6th week of training the rats were running 26.8 meters per minute (2.7 miles per hour) for 90 minutes each day. Then, 34 sedentary and 29 trained rats were given a "progressive work-heat tolerance test" in which the rats were forced to run uphill on a motorized treadmill to avoid electric shock. The room temperature, steepness and speed of the treadmill were increased stepwise "until the animals were exhausted" (refused to run). "Fatalities" were defined as those animals that died within 24 hours. "Survivors"	university	Navy

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were those animals still alive after 24 hours. After 24 hours, blood samples were taken in survivors by heart puncture and analyzed for biochemical changes which are known to indicate heat stroke. Other sedentary and trained rats were used in "control" studies, in which the rats were subjected to either electric shock, forced treadmill running or heat stress, to observe the possible effects of any of these procedures separately on biochemical changes associated with heat stroke. There was no significant difference between sedentary and trained rats in the number of fatalities: 13 trained and 14 sedentary rats died. However, in survivors, trained rats were able to continue running much longer and at significantly higher temperatures than sedentary rats before exhaustion. Blood analysis in all survivors indicated severe heat stroke.		
Non-impact Brain Concussion - 1982 Traumatic coma was produced in 45 monkeys by accelerating their heads without impact. The animals were anesthetized, their heads placed into metal helmets and held rigidly in place by "dental stone." The helmet was attached to an accelerator which shook the monkeys' head either sagittally (forward and backward), laterally (side to side), or obliquely (diagonally), producing traumatic coma of varying duration and severity. Seven of the monkeys which were still in coma after 2 hours were killed for biochemical studies. *Cerebral concussion* (coma of less than 15 minutes) occurred in 11 of the animals whose heads were shaken sagittally, 2 of the obliquely shaken animals, and 2 of the laterally shaken animals. Coma occurred in these animals "at the moment of acceleration" and was accompanied by unconciousness, dilated and unresponsive pupils, lack of corneal reflexes (contraction of the eyelids when the eye is lightly touched), and absent "lash reflexes." All of these animals recovered quickly and with no complications. *Mild prolonged traumatic coma* (unconsciousness lasting longer than 15 minutes but less than 2 hours) occurred in 2 of the sagittally shaken monkeys, 3 of the obliquely shaken monkeys, and 1 laterally shaken monkey. Symptoms of the "initial phase of coma" were identical to those that occurred during concussion; however, recovery was prolonged and incomplete in all monkeys. After the initial phase of coma, which lasted approximately 10 minutes, there was a short period of intermediate coma, during which the animals' corneal reflex returned, but the eyes remained closed and the animals remained unresponsive. Emergence from coma took an average of 75 minutes, during which the animals remained unresponsive but occasionally opened their eyes. Twenty-four hours after injury, the animals were conscious but had "few spontaneous movements [and] could not stand or climb." Behavior and motor activity improved over the next several days so that the animals showed only "moderate disability" (able to eat and drink without assistance); however, none became "normal". *Moderate prolonged traumatic coma* (2-6 hours duration) was seen in 4 animals, 1 in the oblique group and 3 in the lateral group. Initial phase of coma lasted 15 minutes, and intermediate coma was present for an average of 4 hours. Emergence from coma took approximately	university	National Institutes of Health

Description	Type of Institution	Source of Funds
8 hours. Only one animal recovered to "moderate disability." The remaining 3 never recovered sufficiently to eat, drink, or walk. Motor activity in these animals was uncoordinated and often "non-purposeful." They required "total nutritional support" until they were killed for autopsy. *Severe prolonged traumatic coma,* lasting more than 6 hours, occurred in 13 monkeys, all shaken laterally. Initial phase of coma lasted an average of 45 minutes, and intermediate coma was present up to 7 days in some animals. Four of the animals were killed for autopsy without regaining consciousness, and none of the remaining animals recovered enough to eat or drink. "Best recovery was to a state of severe disability necessitating total care." Of the 13 animals with severe prolonged traumatic coma, 12 showed abnormal "posturing" during intermittent coma. Nine of these showed "decerebrate postures" (limbs extended rigidly, as seen in decerebrate animals whose brain has been surgically separated from the body) in all 4 limbs. Two other animals showed decerebrate postures in the lower limbs and in 1 upper limb. The remaining upper limb showed "decorticate postures" (limbs rigidly flexed), as seen in animals whose cerebral cortex (a part of the brain) has been surgically separated from the body. The last animal showed a "crossed pattern" with decerebrate postures (limb extension) in the right upper and left lower limbs and decorticate postures (limb flexion) in the left upper and right lower limbs. Similar postures were also seen in the 7 monkeys which were killed after 2 hours for biochemical studies, indicating that they were also in severe prolonged traumatic coma. Eight weeks after head injury, the remaining animals were killed for autopsy. Examination of their brains showed brain damage, the extent of which correlated with the severity of the animal's coma. The experimenters concluded that brain damage "produced by... head acceleration is a major cause of prolonged traumatic coma."		
Impact - 1983 123 rabbits were anesthetized and subjected to "blunt, nonpenetrating thoracic impact." In 28 rabbits, impact resulted in "sudden death." Surviving animals were observed for 15 minutes after impact, then killed, and all rabbits were autopsied. Injuries included cardiac tamponade (compression of the heart caused by severe hemorrhage), rupture of heart chamber and/or major blood vessels, bruising of the heart muscle and/or major blood vessels, and injury to liver.	private	General Motors
Air Force Plaster Casts Monkeys for 8 Weeks - 1983 In 1983, Air Force Researchers reported the results of an experiment in which four groups of rhesus monkeys were used to observe the effects of immobilization on bones. The first group was immobilized in plaster casts extending from the neck to the ankles for 8 weeks. The second group was also immobilized in plaster casts for 8 weeks; however, either the right or left leg of each monkey was "freely moveable." The free foot was strapped to a pedal which was connected by a pulley to 5-pound weights. When the monkeys "exercised" the free leg by pushing the weighted pedal down "about 12 inches" banana pellets were released into their mouths. The third group	government - military	Air Force

Description	Type of Institution	Source of Funds
was an "untreated" control group. The fourth group was immobilized, but was returned to the "gang cage" after decasting for a "reconditioning period" of either 5 or 12 months. After 8 weeks, the monkeys in the first two groups and the controls were killed, and their bones were removed and analyzed for physiological, biomechanical, and biochemical changes. The animals in the fourth group were killed following the "reconditioning period."		
Mental Retardation - 1983 To observe Pavlovian classical conditioning of heart rate produced by an aversive stimulus, (electric shock), adult cats ("obtained locally" of "unknown history") and kittens were surgically fitted with a "cranial implant," then placed in a harness with their feet on a treadmill. The "cranial implant" was then bolted to a steel frame to immobilize the animals' heads. Rods connected to a measuring device were attached to the cats' left legs to record leg movements. An air blast was delivered to the base of the tail, followed by electric shocks to the pads of the left foot and to the tail. This forced the cats to run on the treadmill while at the same time raising the leg to try to escape the electric shock, and increased the animals' heart rates. After several pairings of electric shock and air blast, the older cats responded with increased heart rate and running in the presence of air blast alone, just as Pavlov's dogs learned to salivate when they heard a bell but got no food. The kittens did not learn to respond to air blast alone.	university	National Institutes of Health
Mental Retardation - 1981 Kittens were injected with PCP (phencyclidine, a popular street drug called "angel dust"). Kittens under three weeks old cried out loudly and crawled along the floor dragging their hindfeet, with their heads down and mouths touching the floor. Sometimes they would crawl in a circle, or even backward. They crawled over obstacles, such as a group of littermates, lying in their path. When they came to a wall, they pushed against it with their heads and forelimbs until they were turned sideways, then continued crawling. Occasionally, the kittens rolled from side-to-side or onto their backs, accompanied by tremors of the hind limbs. In kittens older than three weeks, PCP injection resulted in staggering, collapsing, and "waxy rigidity" in which kittens "placed" in abnormal postures (limbs extended at odd angles) remained that way. Drugged kittens of all ages ignored normal littermates who tried to play with them, and continued their abnormal behavior.	university	U.S. Public Health Service

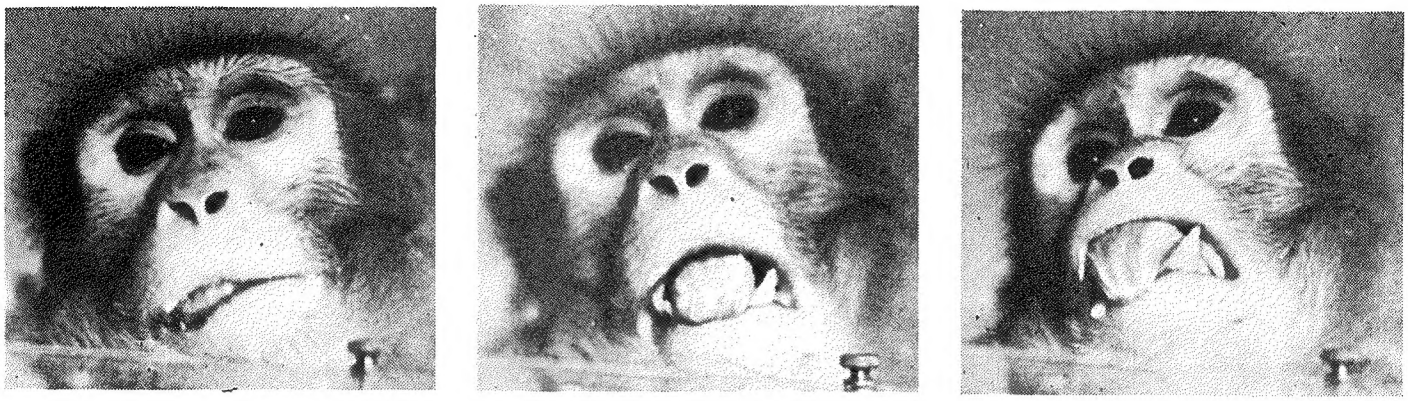
The above examples of animal experiments illustrate, we think, why pompous opinions, a lot of dark innuendo or vague adjectives - or even those tired cliches about "morals and ethics" - are no substitute for reporting the crimes being committed against laboratory animals. Lawyers, legislators, philosophers and other responsible people can't work with obscure generalities.

* * *

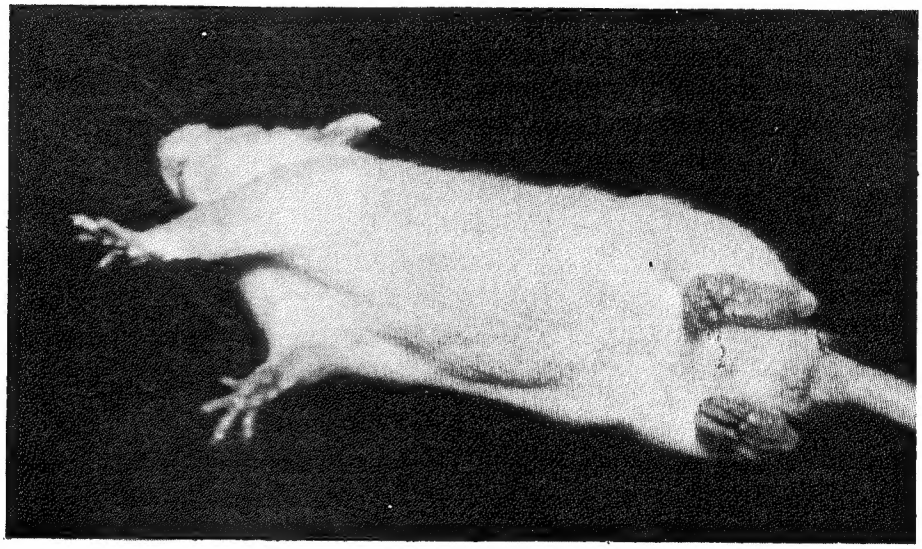
Rigidity due to fear of humans.

Suffering laboratory rat mutilates own tail.

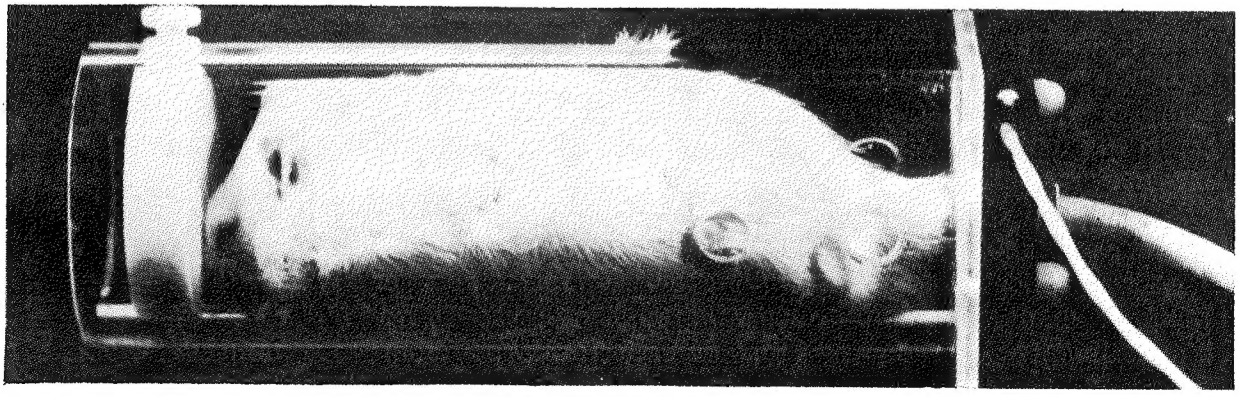
Researcher sexually stimulates brain-lesioned cat.



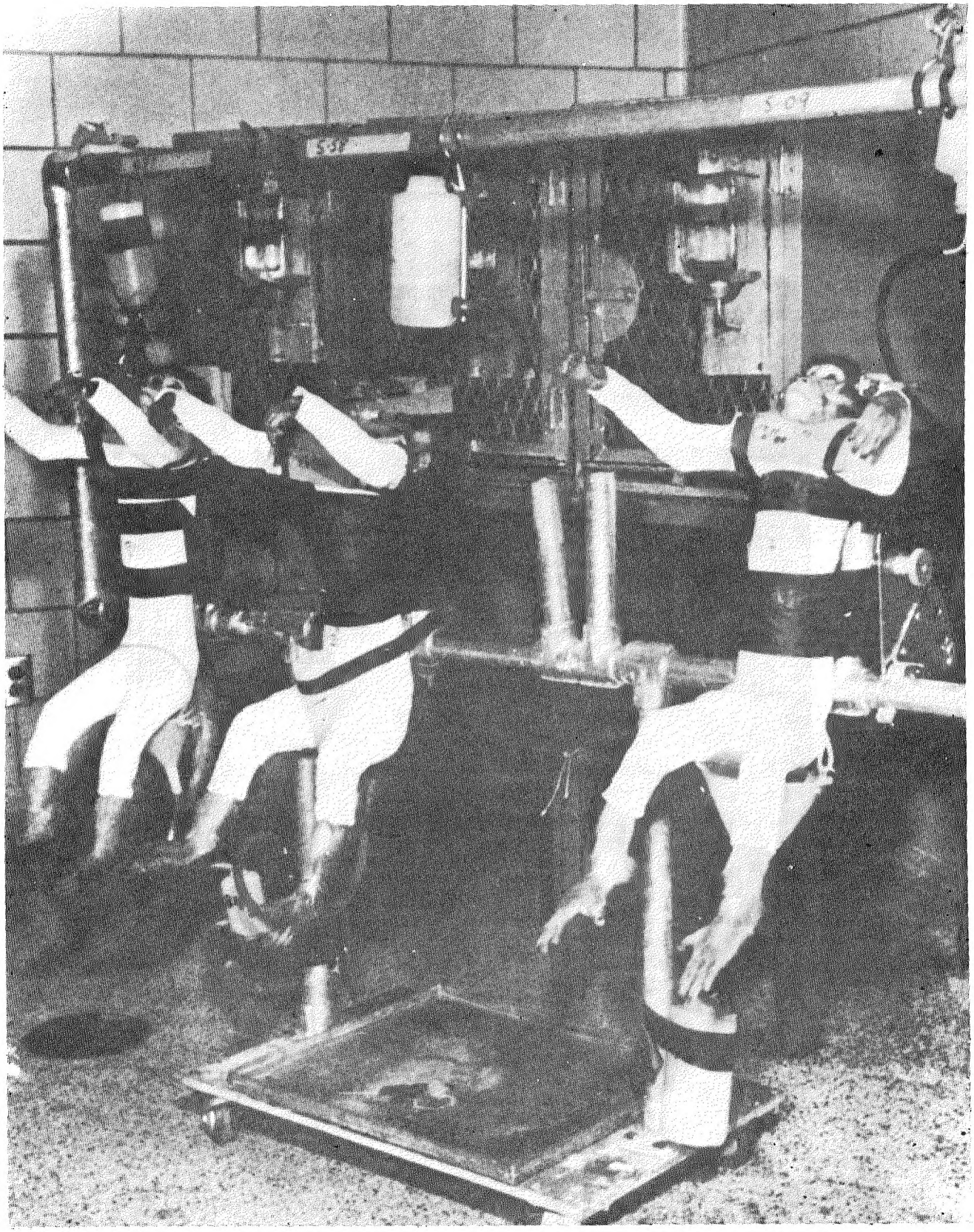
Psycho drug (TD) monkeys



Little laboratory rat convulsing in agony.



Both rat and tube wet from animal's salivating in fear and pain from electric shock to tail.



Air Force emulates NASA: Immobilizes primates in plaster casts for 8 weeks.

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